N-3 Essential Fatty Acids Intake and Depressive Symptoms

Nami Matsumura

Follow this and additional works at: https://commons.und.edu/theses

Part of the Psychology Commons

Recommended Citation

Matsumura, Nami, "N-3 Essential Fatty Acids Intake and Depressive Symptoms" (2000). Theses and Dissertations. 924.
https://commons.und.edu/theses/924
N-3 ESSENTIAL FATTY ACIDS INTAKE AND DEPRESSIVE SYMPTOMS

by

Nami Matsumura

Bachelor of Sciences, High Point University, 1997

A Thesis

Submitted to the Graduate Faculty

of the

University of North Dakota

in partial fulfillment of the requirements

for the degree of

Master of Arts

Grand Forks, North Dakota

May

2000
This thesis, submitted by Nami Matsumura in partial fulfillment of the requirements for the Degree of Master of Arts from the University of North Dakota, has been read by the Faculty Advisory Committee under whom the work has been done and is hereby approved.

[Signature]
(Chairperson)

This thesis meets the standards for appearance, conforms to the style and format requirements of the Graduate School of the University of North Dakota, and is hereby approved.

[Signature]
(Dean of the Graduate School)

4-30-00
(Date)
PERMISSION

Title N-3 Essential Fatty Acids Intake and Depressive Symptoms

Department Counseling

Degree Master of Arts

In presenting this thesis in partial fulfillment of the requirements for a graduate degree from the University of North Dakota, I agree that the library of this University shall make it freely available for inspection. I further agree that permission for extensive copying for scholarly purposes may be granted by the professor who supervised my thesis work or, in her absence, by the chairperson of the department or the dean of the Graduate School. It is understood that any copying or publication or other use of this thesis or part thereof for financial gain shall not be allowed without my written permission. It is also understood that due recognition shall be given to me and to the University of North Dakota in any scholarly use which may be made of any material in my thesis.

Signature Nami Matsumura
Date 4/27/00
<table>
<thead>
<tr>
<th>TABLE OF CONTENTS</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>LIST OF TABLES......................................................</td>
<td>vi</td>
</tr>
<tr>
<td>ACKNOWLEDGMENTS......................................................</td>
<td>vii</td>
</tr>
<tr>
<td>ABSTRACT...........................................................................</td>
<td>viii</td>
</tr>
<tr>
<td>CHAPTER...........................................................................</td>
<td></td>
</tr>
<tr>
<td>I. INTRODUCTION...........................................................</td>
<td>1</td>
</tr>
<tr>
<td>II. LITERATURE REVIEW..................................................</td>
<td>8</td>
</tr>
<tr>
<td>Possible Relationships between Poor Diets and Depression</td>
<td>8</td>
</tr>
<tr>
<td>Lowering Cholesterol...............................................</td>
<td>8</td>
</tr>
<tr>
<td>Age Related Differences..........................................</td>
<td>9</td>
</tr>
<tr>
<td>Postpartum and Suicides...........................................</td>
<td>15</td>
</tr>
<tr>
<td>Fatty Acids.............................................................</td>
<td>17</td>
</tr>
<tr>
<td>Polyunsaturated Fatty Acids.....................................</td>
<td>17</td>
</tr>
<tr>
<td>American Diet and Depression...................................</td>
<td>19</td>
</tr>
<tr>
<td>Explanations for the Connection...............................</td>
<td>22</td>
</tr>
<tr>
<td>Effects of N-3: True or False?..................................</td>
<td>23</td>
</tr>
<tr>
<td>Summary.........................................................................</td>
<td>25</td>
</tr>
<tr>
<td>III. METHODOLOGY..........................................................</td>
<td>27</td>
</tr>
<tr>
<td>Research Question...................................................</td>
<td>27</td>
</tr>
<tr>
<td>Data...............................................................................</td>
<td>27</td>
</tr>
<tr>
<td>Data Analysis..........................................................</td>
<td>30</td>
</tr>
</tbody>
</table>
### LIST OF TABLES

<table>
<thead>
<tr>
<th>Table</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>2. Population Estimates for the US., by Regions, and States by Selected Age Group and Sex: July 1, 1990</td>
<td>43</td>
</tr>
<tr>
<td>3. Food Intakes by Simmons Market Research Bureau, Inc. 1994</td>
<td>44</td>
</tr>
</tbody>
</table>
ACKNOWLEDGMENTS

I wish to express sincere appreciation to my thesis advisor, Dr. George Henly, for his time, encouragement, and valuable recommendations in the development and completion of this thesis. I gratefully acknowledge my committee members, Dr. Cindy Juntunen and Dr. Sue Jacobs, for their time, and helpful comments and suggestions. I would like to thank Ms. Jan Goodwin, for her time, and helpful comments and suggestions in regards to a specific aspect—nutrition—of my paper. I also wish to acknowledge and thank Greg Tierney for assisting me in analyzing the data.
ABSTRACT

The purpose of this study was to examine whether N-3 fatty acids intake would correlate with a prevalence of mental distresses. The present study consisted of a comprehensive literature review and analysis of preexisting data that were relevant to this study. Two data were primarily used in the assessment. These were the 1994 study of Media and Markets by Simmons Market Research Bureau, Inc. for food intakes, and the report of the Behavioral Risk Factor Surveillance System (BRFSS) for the prevalence of Frequent Mental Distress. Due to incompatibility of those two studies, additional data—the U.S. population estimates for regions and states by selected age and sex group: annual series, July 1, 1990—was included in order to make the first two data comparable.

The data were independently analyzed for more sensitive analysis. From the descriptive results, the overall regional prevalence of Frequent Mental Distress was the lowest in the Midwest and the highest in the West. The South region that exhibited the second lowest prevalence of mental distress showed the highest consumption of fishes and vegetable oils that were the source of N-3, and the West which exhibited the highest prevalence of mental distress showed the lowest in food intakes. The analysis of mental distress based on age-sex groups showed significant differences among regions except one age-sex group (Male aged 45 to 64 years). Thus, there were significant differences among regions in terms of the prevalence of mental distress, but I could not make a conclusion that the food consumptions might contribute to the regional differences in the
prevalence of frequent mental distress because there were no correlation between them observed in this study.
CHAPTER I
INTRODUCTION

The human body needs a certain type and amount of nutrients in order to maintain itself and to function adequately and effectively. For example, the very tissues of our bodies, the fuels for the cells, and the hormones that regulate our body must be furnished by the foods we eat. The foods we eat have measurable effects on the body's performance, including both physical and mental capabilities. “The idea that the right foods, or the natural neurochemicals they contain, can enhance mental capabilities—help you concentrate, tune sensorimotor skills, keep you motivated, magnify memory, speed reaction times, defuse stress, perhaps even prevent brain aging—is not idle speculation” (Blaun, 1996, p. 35). In addition, Schafer, Keith, and Schafer (1994) have stated that a nutritionally healthy diet makes us capable to behave adequately and effectively. Thus, it is important for us to eat nutritious foods. Unhealthy diets are likely to negatively affect our body's performance. This means that the unhealthy diets may affect our mood or may have some other negative psychological effects on us.

Depression is a major and common psychological problem. It can vary enormously in its duration and intensity, but almost everyone has had at least an occasional, brief period of feeling sad and discouraged. Just as people experience occasional anxiety, they will probably have more than an ample amount of sadness during the course of their lives (Neale & Davidson, 1994). Depression is a major concern
for American people because of high rates of depression among them. According to a recent study by the World Health Organization, the World Bank, and Harvard University (1999), more than 19 million adult Americans aged 18 and over will suffer from a depressive illness—major depression, bipolar disorder, or dysthymia—each year. Compared to Asian countries, such as Japan and Taiwan, the North American population showed cumulative rates of depression 10-fold greater in an intensive cross-national collaborative study of rates of depression (Hibbeln & Salem, 1995).

Depression is an emotional state marked by great sadness and apprehension, feelings of worthlessness and sometimes, guilt, withdrawal from others, loss of sleep, appetite, sexual desire, and loss of interests and pleasure in usual activities (Neale & Davidson, 1994). Depressed persons feel fearful and gloomy, helpless, and hopeless. They may feel everything is out of their control. Their lives seem unhappy and meaningless. As a consequence, they may lose their appetites.

Poor diets and depression should not be considered as separate phenomena, because they are highly correlated and may influence each other. The best example of the relationship between poor diets and depression would be eating disorders, such as anorexia and bulimia nervosa. Eckerd, Goldberg, Halmi, Casper, and Davis (as cited in Neale & Davidson, 1994) found high levels of depression among 105 hospitalized anorexics, especially in those with the most severe eating difficulties. According to Kaye, Weltzin, and Hsu (1993), 21% to 91% of anorexic patients have depressive symptoms when they are underweight and malnourished. In these disorders, depression plays an important role, not just as an understandable consequence but as a cause as well.
Although depressed people are more likely to lose their appetite as a consequence of depression, anorexic persons are so afraid of becoming fat that they refuse to eat. As a consequence, their bodies become weaker and weaker. If anorexics lose more than 25 percent of their body weight, the body temperature will subsequently drop and the consequence might be starvation, which causes biochemical changes that bring on depression (Neale & Davidson, 1994). Bulimia nervosa is another eating disorder that can cause similar harmful somatic changes on their bodies. Unlike anorexics, bulimics eat, and sometimes overeat, food. However, their bodies are not nourished by the food they eat because they induce vomiting or overdoses of laxatives to get rid of food they just ate. As a result, their bodies experienced somatic changes similar to those of anorexics. A poor diet causes a biochemical change at the synapse, and that change might cause depression. As a matter of fact, underweight anorexics have reduced serotonin activity (Kaye, Weltzin, & Hse, 1993). It is not clear whether depression or unusual eating habits (poor diets) come first in eating disorders, but it is clear that the changes in physiological chemicals, such as neurotransmitters, play an important role in emotional states.

Another example of the relationship between poor diets and depression is suicides. Depression is a risk factor for suicides and fatal accidents, in which depression may relate to low cholesterol (Morgan, Palinkas, Barrett-Connor, & Wingard, 1993). There may be an association between low serum cholesterol and suicide, but it is unclear whether low serum cholesterol level or depression comes first in suicides. The association may arise because low serum cholesterol causes depression, or because it is a consequence of depression (Law, 1996). Depressed people simply eat less and as a result,
their bodies are not nourished by food. In both cases of eating disorders and suicides, unusual or irregular eating habits may cause changes in physiological chemicals, such as neurotransmitters that play an important role in people’s emotional states.

According to Kalat (1995), a neurotransmitter is a basic chemical that affects both physiological mechanism and emotional states. It is released at a synapse (a point of communication between two neurons or between a neuron and a muscle). Each neuron releases the same combination of neurotransmitters from all branches of its axon. At certain synapses, a neurotransmitter exerts its effects by attaching to a receptor that opens the gates to allow a particular ion, such as sodium, to cross the membrane more readily. At other synapses, a neurotransmitter may lead to slower but longer-lasting changes inside the postsynaptic cell. Activation of such receptors may inhibit or facilitate the release of a neurotransmitter from the axon. After the activation, some of the transmitter molecules are reabsorbed and eventually excreted. Different neurotransmitters contribute to behavior in different ways. Therefore, certain behavioral abnormalities can be traced to an excess or deficit of chemical activity at particular types of synapses.

The amount of neurotransmitters can be considered as a primary cause of depression although the mechanisms of depression are more complex than simply having too much or too little of a particular neurotransmitter. There are two neurotransmitters—norepinephrine and serotonin—that probably explain a relationship between food and depression. Norepinephrine and serotonin are chemicals believed to act as neurotransmitters at various places in the nervous system. “Considerable data in experimental animals and clinical studies in other psychiatric patient groups indicate that
these two neurotransmitter systems contribute to the modulation of appetite, mood, personality variables, and neuroendocrine function” (Kaye, Weltzin, & Hsu, 1993, p. 127).

One theory involving norepinephrine (NE) posits that a low level of norepinephrine leads to depression and a high level leads to mania. When administered into specific areas of the hypothalamus involved in feeding modulation, NE, at near physiological doses, produces eating in satiated animals (Kaye, Weltzin, & Hsu, 1993). The less NE is produced in the specific area of hypothalamus, the less animals or people feel hungry. The other theory, involving serotonin, suggests that a low level of serotonin, which frequently serves to regulate neural activity in other neurochemical systems, allows wild fluctuations in the activity of other neurotransmitters, thereby producing both mania and depression (Kalat, 1995). Lowering serum cholesterol concentration causes changes in the cholesterol content of the synaptosomal membrane and a decrease in the number of serotonin receptors. Reduced serum cholesterol concentrations lower neuronal serotonin release, thereby precipitating the abnormal behaviors, such as suicidal behavior and impulsive aggression (Fernstrom, Verrico, Ebaugh, & Fernstrom, 1996). Because a low serotonin concentration has been associated with suicidal depression and impulsive behavior, lowered serotonin concentration or fewer serotonin receptors may account for the increase in deaths from external causes, such as suicides, in the treatment groups in cholesterol lowering trials (Brown, Salive, Harris, Buralnik, & Kohout, 1994). The amount of neurotransmitters is affected by our serum cholesterol consumption. The more we consume cholesterol, the more neurotransmitters will be produced, and vice versa.
Nutritional status can affect both peripheral and central noradrenergic function (Kaye, Weltzin, & Hsu, 1993). A poor diet can lead to physiological changes in our bodies and may cause depression. There are certain kinds of nutrients thought of as a link with depression. These are cholesterol and fatty acids, especially polyunsaturated fatty acids.

There is little research on the correlation between food and depression. However, some studies show that there may be a relationship between depression and serum cholesterol or polyunsaturated fatty acids. The serum cholesterol level, especially, has been recognized as a link with depression (Law, 1996), and cholesterol is highly correlated with fatty acids in diets. Polyunsaturated fatty acid is the main focus in my study, but I will also discuss the possible association between serum cholesterol and depression because cholesterol and fatty acids are obtained from same or similar food.

The long chain polyunsaturated (LCP) fatty acids are obtained either from the diet or by elongation and desaturation of LCP such as linoleic (n-6) and linolenic (n-3) acids. These two parent fatty acids are called the essential fatty acids because they cannot be manufactured in the body; thus, we need to obtain those from the food we eat. N-3 fatty acids are a primary focus of my study since they seem to correlate with depression (Rudin & Felix, 1987, 1996). N-3 fatty acids can be obtained from such fish as mackerel, sardines, tuna, trout, and cold water salmon (Finnegan, 1992) and such vegetable oils as flax seed, canola, soybean, and walnut oils (Hibbeln & Salem, 1995).

The goal of this study will be to assess the correlation between the consumption of certain vegetable oils or fish that include n-3 fatty acids and persons’ affective states. My hypothesis is that people who take in an optimal level of serum cholesterol and fatty
acids (which can be determined by a person’s body weight and height according to Finnegan [1992]) will feel less depressed than people with too much or too little of those nutrients.
Possible Relationships between Poor Diets and Depression

Lowering Cholesterol

In the United States, a lower cholesterol diet is highly recommended due to high rates of cardiovascular diseases and obesity. These two disorders are major problem in the U.S. Currently, about one of three adults is currently overweight based on self-reported height and weight, compared with about one of five in the late 1970’s (Enns, Borrud, Mickle, Chahil, & Fowler, 1998). A high level of cholesterol is not good for our health because it increases the risk of heart disease and death. In fact, heart disease is the number one killer in the United States (Stowell, 2000). Reducing serum cholesterol has been considered and recognized as a desirable goal for a long time, particularly for those with high cholesterol concentrations and a history of atherosclerotic disease (Brown, 1996). Cholesterol lowering treatment (reducing 20-40%) seems to be effective and thus, current guidelines recommend a treatment goal for low density lipoprotein cholesterol (LDL) of 2.6 mmol/L (mmol/L = mg/dL*0.02586) in patients with coronary disease (Rosengren, 1998). However, it is important to keep in mind in a cholesterol reduction program that cholesterol levels increase with age and are slightly higher in males than females. For example, typical mean levels of cholesterol at age 40-44 are 5.3 mmol/L for males and 5.0 mmol/L for females (Boston, Dursun, & Reveley, 1996). Without
considering age and gender differences in cholesterol levels, the diet program may cause harm to our bodies.

Several researchers have suggested that encouraging a lower cholesterol diet may produce some harm to our bodies as well as to our emotional states. A high serum cholesterol concentration is a well established risk factor for heart disease, but the results of cholesterol reducing clinical trials have suggested that reducing cholesterol concentration might increase deaths from external causes such as suicide, accidents, and murder (Brown et al., 1994). A meta-analysis of six primary prevention trials suggested that coronary heart disease mortality tended to be lower in men who were treated with a cholesterol lowering regimen, but it showed the possibility that lowering cholesterol might contribute to the violent deaths (Brown, 1996). The following studies investigated the possible relationships between reduced cholesterol levels and depression or suicide.

**Age Related Differences**

The first four studies involved age considerations. One cohort study was designed by Weidner, Conner, Hollis, and Conner (1992) in order to measure changes in negative emotions in relation to diet and plasma cholesterol levels. They administered assessments before and after a 5-year dietary intervention program, the Family Heart Survey, aimed at reducing plasma cholesterol levels. The hypothesis was that reducing dietary and plasma cholesterol might lead to more negative emotions and might contribute to an increment of suicide and violent deaths. Participants were 149 men and 156 women from 233 families.
The mean age of these participants was 37.7 years. These families were selected randomly from 3579 homes in a representative urban, middle class neighborhood in Portland, Oregon. In this study, an eating habit questionnaire was used, that yielded four dietary categories: 37% of calories from fat, 30% from fat, 25% from fat, and 20% from fat. Participants were assigned to a dietary category, based on eating behaviors that reflected intake of total and saturated fat, cholesterol, and complex carbohydrates. The Hopkins Symptom Checklist (SCL-90) was used to assess depression and aggressive hostility as well as seven other dimensions of negative emotions (anxiety, somatization, phobic anxiety, paranoid ideation, interpersonal sensitivity, obsessive-compulsive behavior, and psychoticism).

The results indicated that those who consumed a low-fat (≤ 30% kcal from fat), high complex-carbohydrate diet at the end of the study showed significantly greater improvement in depression as well as a reduction in their plasma cholesterol levels compared with those who were on a high-fat ‘American diet (37% kcal from fat).’

Weidner et al. (1992) found that improvements in diet were associated with reduced levels of depression. However, they did not find evidences to support their hypothesis that reducing dietary and plasma cholesterol might lead to a more negative emotions and contribute to an increment of suicide because the cholesterol-lowering program did not make the depressed participants feel worse in emotional states. The important point from this study was that eating healthy food might improve a person’s emotional states and cholesterol consumption level was associated with emotional states in their sample. This
study was based on the middle-aged sample. The following were based on elderly samples.

The elderly have markedly higher rates of depression than younger people, despite the fact that elderly people have more cholesterol than younger people because cholesterol concentration levels increase with age. The elderly might reduce cholesterol and other nutrients believing that they don’t need to eat much because their activity levels go down with age. Whatever the reasons are, some elderly experience malnutrious conditions. “Overall, 5 % to 15 % of elderly adults may have nutrient deficiencies” (ADA, 1993 cited in Dudek, 1997). The idea that lowing cholesterol and fats is healthy might overwhelm the elderly population as well as other populations. It might not be necessary for the elderly to reduce cholesterol because serum cholesterol levels as a risk factor for chronic heart disease become less important after age 64 (Dudek, 1997). In addition, lowering fat and saturated fat may not be appropriate for older adults who are at risk for malnutrition because of the potential negative impact on overall calorie and nutrient intake. Also, “overly-restrictive diets may actually contribute to malnutrition” (Administration on Aging, DHHS, 1994, p.357, cited in Dudek, 1997).

One study to assess the correlation between age and low cholesterol level in depression was designed by Morgan, Palinkas, Barrett-Connor, and Wingard (1993). They carried out a population-based study to investigate whether depressive illness was related to low plasma cholesterol concentrations in men of 50 years and older. Participants were 1020 white men, all adult residents of Rancho Bernardo, California. The study was executed through three stages. At the first stage, between 1972 and 1974,
82% of the participants took part in a survey of risk factors for heart disease as a part of Lipid Research Clinic prevalence study. At the second stage, held in 1984, all surviving participants—more than 79% of the original participants—took part in a follow-up study designed primarily to screen for diabetes and other chronic diseases. At the final stage, held in 1987, their depressed mood was assessed on the basis of response to eighteen items of the Beck depression inventory (BDI).

Morgan et al. (1993) found that depressive symptoms increased significantly with age, and lower mean plasma cholesterol concentration was associated with age. "Higher depressive symptom scores and the prevalence of categorically defined depression were associated with cholesterol values below 4.14 mmol/L in men 70 years and older" (Morgan et al., 1993, p. 77). A cholesterol concentration below 4.14 mmol/L was associated with a 16% prevalence of categorical depression in men 70 years and older. Therefore, Morgan et al. (1993) concluded that the elderly were more likely to become depressed due to having lower cholesterol levels than younger people. Although the cholesterol level contributed less than age, health, and level of physical functioning, low cholesterol (<4.14 v.s. ≥ 4.14 mmol/L) remained a significant independent predictor of depression.

Another study to assess the correlation between age and low cholesterol level in depression was designed by Brown, Salive, Harris, Simonsick, Guralnik, and Kohout (1994). They investigated the association between low serum cholesterol concentration and severe depressive symptoms in an elderly population. The design was a cross sectional analysis of pooled data from three communities of the established populations.
for epidemiologic studies of the elderly. Participants who completed their interview, the Centers for Epidemiologic Studies' Depression Scale, and consented to measurement of their cholesterol concentration were included in the study. The participants were 3939 women and men aged 71 or older. Chi Square ($X^2$) analyses, $t$ tests, and multivariate regression analyses were used to measure the association between low cholesterol concentration and severe depressive symptoms. All analyses were stratified by sex, and multivariate analyses were adjusted for age, self-reported health, physical function, number of drugs used, and weight loss.

The result of Brown et al. (1994) showed depressive symptoms were higher in the older subjects ($\geq 80$) than in younger ones ($< 80$) in both men and women. The older age group had a significantly lower score in mean cholesterol concentration. Men and women aged $\geq 80$ with a low cholesterol concentration were nearly twice as likely to have severe depressive symptoms as those with a normal or high concentration. Depressive symptoms, cholesterol concentration, weight, and use of drugs were all associated with age in both men and women. Their result was compatible with the conclusion made by Morgan et al (1993). However, in multivariate models that adjusted for age, self-reported health, physical function, number of drugs used, and weight loss, the association was substantially weakened. This means that low cholesterol concentration was not associated with severe depressive symptoms in this age group after other factors were controlled for. Therefore, Brown et al. (1994) could not conclude that low cholesterol could be a cause for depression because no statistically significant association between low cholesterol
concentration and severe depressive symptoms was found, although an association between low cholesterol and depression could be seen in the elderly men and women.

The other study to assess the correlation between age and a low serum cholesterol level in depression was designed by Rozzini, Bertozzi, Barbisoni, and Trabucchi (1996). Data were obtained from 476 elderly patients (mean age 78.8 years; 70% women) admitted to their geriatric evaluation and rehabilitation unit over 12 months. They assessed with a multidimensional evaluation, including demographic information, physical health, cognitive status, functional ability, and social support. These patients were divided into three groups of decreasing serum cholesterol concentrations: ≥ 6.20, 4.14-6.19, and ≤ 4.13 mmol/L. The association of serum cholesterol groups with depression was tested in a multiple logistic regression model in which confounding by age, sex, and other factors was controlled. The result showed that the risk of depression was greater in the group with lowest serum cholesterol concentration even after the potential confounding factors were considered. However, there was no statistical significance obtained in this study. Therefore, Rozzini et al. (1996) could not support their hypothesis, although they argued that it might be clinically relevant in elderly people.

All these studies have shown that there may be some relationship between depression and lowering cholesterol, especially among elderly people. Two studies found a significant association, but the other two studies found no significant association between depressive symptoms and lowering cholesterol. One explanation for this relationship may be that lowering cholesterol concentration causes changes in the
cholesterol content of the synaptosomal membrane and decreases in the number of serotonin receptors. Decreasing the level of serotonin allows wild fluctuations in the activity of other neurotransmitters, thereby producing both mania and depression. A low serotonin concentration has been associated with suicidal depression and impulsive behavior; thus, lowered serotonin concentration or perhaps fewer serotonin receptors could be a cause for deaths from external causes in the treatment group in cholesterol lowering trials (Brown et al., 1994). All these four studies found some links between lower cholesterol and depression, but they needed further research to explain the association between them.

Postpartum and Suicides

Two other studies shed further light on the puzzling issues of cholesterol concentrations and depressive symptoms or suicide. The first was a cohort study, conducted by Zureik, Courbon, & Ducimetiere (1996) with annual repeated measurements of serum cholesterol concentration in order to investigate whether low serum cholesterol concentration or changing it is associated with risk of suicide in men. The participants were 6393 working men (aged 43 to 52) in Paris, France between 1967 and 1972. They measured individual change in serum cholesterol concentration and death from suicide three times during the study period as well as during average of 17 years’ follow up.

The relative risk of suicide for men with low average serum cholesterol concentration (< 4.78 mmol/L) was compared with that for those with average serum cholesterol concentration of 4.78 to 6.21 mmol/L. The result indicated that men whose
serum cholesterol concentration decreased by more than 0.13 mmol/L a year had higher multivariate adjusted relative risk than those who remained stable. Low serum cholesterol concentration increased risk of death from suicide in men. However, long term surveillance of subjects included in trials of lipid lowering treatments seems warranted and needs further investigation.

The second study was conducted by Ploeckinger, Dantendorfer, Ulm, Baischer, Derfler, Musalek and Dadak (1996). They used pregnancy as a natural model for the effect on mood of a reduction in cholesterol because the rapid physiological drop in serum cholesterol in pregnancy was considered to be compatible with postpartum cholesterol levels. Both pregnancy and postpartum lead to a depletion of maternal plasma docosahexaehoic acid (DHA) that is ingested directly or biosynthesized from essential fatty acid precursors (Hibbeln & Salem, 1995). Participants were 20 healthy primiparous women (mean age 25.3 years). A structured clinical interview was held by a psychiatrist, based on the Diagnostic and Statistical Manual of Mental Disorders, third edition. Serum concentrations of total cholesterol and other biochemicals were measured after overnight fasting at two weeks before the expected delivery and at the first and third days after the deliveries of those participants. They used the Zung self rating depression scale to assess mood and Pearson correlation coefficients between mood and absolute lipid concentrations and relative changes in serum lipids to analyze the obtained data. The absolute postpartum cholesterol concentration was not associated with depressive symptom scores, but relative decrease in serum cholesterol from two weeks prepartum was significantly correlated with postpartum depressive scores.
The findings in these two separate studies also suggested a relationship between lowered serum cholesterol and depressive symptoms or suicides similar to the four studies described previously. Therefore, there might be relationships between low serum cholesterol levels and depression or suicides.

**Fatty Acids**

High cholesterol levels are not only bad for the heart, but also they are dreadful for the brain. High blood levels of cholesterol, and especially of a related saturated blood fat known as triglyceride, are strongly correlated, in both adults and children, with the incidence of affective disorders, including depression, manic depression, and schizoaffective disorder. Most fats in food are composed of triglycerides, which are broken down into various fatty acids. “The more you lower the triglycerides, the more you alleviate the depression” (Blaun, 1996, p. 37).

**Polyunsaturated Fatty Acids**

Previous studies by Fowkes, et al. (1992); Endelberg; and Muldoon et al. (1990, as cited in Hibbeln & Salem, 1995) have both offered and contested the proposition that lowering plasma cholesterol by diet increases depression. Cholesterol is correlated with fatty acids in the diets; thus, polyunsaturated fatty acids may also relate to depression. The study on rats done by Fickova, Hubert, Cremel, & Leray (1998) suggested that the n-3 diet that included (n-3) polyunsaturated fatty acids (32.4 mol/100 mol) in diet might contribute to low concentrations of serum triglycerides, cholesterol and insulin. This study might not be applicable to humans, but it gave an indication of the existence of such an association between depression and n-3 fatty acids.
Polyunsaturated fatty acid is one kind of fatty acid. The long polyunsaturated (LCP) fatty acids are obtained either directly from the diet or by elongation and desaturation of linoleic (n-3) and linolenic (n-6) acids. These two parent fatty acids are called the essential fatty acids because they cannot be manufactured in the body. Thus, they must be supplied by food. LCP are produced primarily in the liver and probably transported in the plasma choline phosphoglycerides (CPG) to the brain. The brain contains considerable amounts of LCP associated with membrane phospholipids (Kalat, 1995).

The linoleic (n-6) and linolenic (n-3) acids are both super unsaturated fats. Both acids have been known to be necessary for normal growth and dermal function (Holman, 1998). N-3 fatty acids are known to be particularly crucial for proper development of the human brain, both before birth and in infancy (Kalat, 1995). Essential fatty acids—particularly n-3s—are well studied for optimal brain function. The quantity and distribution of dietary n-6 and n-3 polyunsaturated essential fatty acids influence serum lipids and alter the biophysical and biochemical properties of cell membranes (Hibbeln & Salem, 1995). In addition, these acids have an inverse correlation with each other (Holman, 1998). This means that the amount of one acid affects those of the other in our bodies. Therefore, we need to watch what we eat on a daily basis.

Decreased n-3 fatty acid consumption correlates with increased rates of depression. Long-chain n-3 polyunsaturated deficiency may also contribute to depressive symptoms in alcoholism, multiple sclerosis, and postpartum depression. Adequate long-chain polyunsaturated fatty acids, particularly docosahexaenoic acid, may reduce the
development of depression just as n-3 polyunsaturated fatty acids may reduce coronary artery disease (Hibbeln & Salem, 1995). In addition, mammals cannot interchange fatty acids between n-6 and n-3 families. We must take those nutrients from food. Although n-3 fatty acids can be replaced by n-6 acids in n-3 deficiency states, biophysical properties may not be fully compensated (Holman, 1998). If people do not take enough n-3 fatty acids from their diets, they are more likely to become depressed. There are two important examples about eating habits and the rate of depression as described next.

American Diet and Depression

One example concerns American diet and higher rates of depression among American people. The present American diet is lacking n-3 fatty acids. Instead, the proportion of n-6 polyunsaturated in the diet has markedly increased. “Fat content ranged from 40 percent of calories—approximating that of the average American—to 10 percent of calories” (Blaun, 1996, p. 38). “During human evolution, ratios of dietary n-6 to n-3 fatty acids were 1:1, but are now estimated to be 10:1 or 25:1” (Hibbeln & Salem, 1995, p. 2). Most people who are eating a typical diet take 25 times as much n-6 fatty acids as n-3 fatty acids. Therefore, the people with typical American diet (more fat and less vegetables) are more likely to become depressed.

Another example is about higher fish consumption and lower rates of depression. Societies consuming large amounts of fish and n-3 fatty acids seem to have lower rates of major depression. For example, Japan, where fish consumption is high, has lower rates of depression. In three elderly Japanese populations, prevalence rates of depressive symptoms have been reported as 1.9% (Hibbeln & Salem, 1995). North American and
European populations showed cumulative rates of depression 10-fold greater than the Taiwanese population in which fish consumption is high like Japan (Hibbeln & Salem, 1995). According to the Epidemiologic Catchment Area study survey (1990), in any 6-month period, 19.5% of adult population of the U.S. (age 18 years and older) suffers from a diagnosable mental disorder; among those disorders, depressive disorders, such as bipolar, major depression, and dysthymia, were suffered by 8.3 percent.

A diet that draws heavily on fatty foods, especially n-6 fatty acids, is harmful for our heart and is linked to cancers as well as being a major correlate of depression as shown in those examples. Three studies have been done in order to assess the relationship between polyunsaturated fatty acids and depression.

One study to assess the relationship between polyunsaturated fatty acids and depression was designed by Ellis and Sanders (1977). It suggested that changes in the fatty acid composition of membrane phospholipids could alter the physiological and functional characteristics of the central nervous system, which in turn could manifest itself as a psychiatric disorder. They investigated whether there was any difference in the long chain polyunsaturated fatty acids (LCP) metabolism of subjects suffering from endogenous depression compared with normal healthy subjects. Fasting blood samples were taken from six subjects with endogenous depression and from six age- and sex-matched healthy controls who were hospital staff. These samples were then compared. They found a striking difference in the fatty acids composition of the plasma choline phosphoglycerides (CPG) in all the subjects with endogenous depression compared to normal subjects. The proportion of LCP was significantly greater in the subjects with
endogenous depression. In particular, the proportions of the n-6 fatty acid, metabolites, eicosapentaenoic acid and decosahexaenic acid were significantly greater in depressive subjects. The increased proportion of LCP in the plasma CPG of the depressed subjects could have an endogenous origin. The increased proportion of these acids could represent a loss of LCP from the brain into the plasma; it is, therefore, possible that the change observed could be directly related to the depressive disorder. Thus, the role of diet cannot be excluded, as it has been shown that the LCP of the plasma and erythrocytes can be altered by diet.

Fehily, Bowey, Ellis, and Meade (1981) started their investigation of the long chain polyunsaturated (LCP) based on the preliminary study by Ellis and Sanders (1977). The participants were 26 endogenous depressed, 23 reactive depressed, 11 other psychiatric disorders, and age- and sex-matched healthy controls were studied. Diagnosis of endogenous or reactive depression was based on clinical interview; patients of doubtful classification were excluded from the study. Some of these patients were drug-free for at least two weeks before the study; the others were receiving a hypnotic and/or a tranquilizer to control for the effects of drugs. Then, these patients participated in an overnight fast. From the result, “the proportion of total LCP was significantly higher in the patients suffering from endogenous depression than in the age- and sex- matched controls” (Fehily et al., 1981, p. 38). Their finding was consistent with the preliminary study by Ellis and Sanders (1977).

Edwards, Peet, Shay, and Horrobin (1998) investigated the association between Omega-3 polyunsaturated fatty acid (PUFA) levels in diet and in red blood cell (RBC)
membranes of depressed patients. The participants were 10 depressed patients and 14 matched healthy controls. They measured RBC membrane levels and dietary PUFAs. Depressed subjects showed a significant depletion of RBC membrane n-3 PUFAs, which was not due to reduced calorie intake. Severity of depression correlated negatively with RBC membrane levels and with dietary intake of n-3 PUFAs. Therefore, they concluded that lower RBC membrane n-3 PUFAs are associated with the severity of depression.

Explanations for the Connection

Dietary long-chain polyunsaturated fatty acids are critical to both nervous system and cardiovascular function. These may be an important confounding factor in the recent observation that lowering serum cholesterol may increase the risk of depression. Following dietary advice to lower serum cholesterol, n-6 polyunsaturated fat is substituted for saturated fat, which increases the ratio of n-6 to n-3 fatty acids, thereby lowering n-3 fatty acids in tissues and organs. A diet reduces serotonergic activities, such as neurotransmitter production, degradation, release, reuptake, and binding. Data concerning the specific effects of dietary long-chain n-3 polyunsaturated fatty acids on biogenic amine function are sparse, but studies of n-3 and n-6 fatty acids do suggest that dietary polyunsaturates may affect noradrenergic and serotonergic neurotransmission (Hibbeln & Salem, 1995). Because serotonin and norepinephrine tend to contribute to the modulation of appetite, mood, personality variables, and neuroendocrine function, disturbances of these neurotransmitter activities could be a factor for depression (Kaye, Weltzin, & Hsu, 1993). The deficits in essential fatty acids can affect the mechanism of neurotransmitters at the synapse, and that change may cause depression.
One mechanism to explain a cause of depression may be a disruption of the biophysical properties of neuronal membranes, which are critically determined by long-chain polyunsaturated fatty acid composition. "Biological properties of synaptic membranes directly influence neurotransmitter biosynthesis, signal transaction, uptake of serotonin binding of adrenergic and serotonergic receptors, and monoamine oxidase activity, factors that are all implicated in the neurobiology of depression" (Hibbeln & Salem, 1995, p. 6). Therefore, inadequate n-3 in the nervous system may increase vulnerability to depression.

The Effects of N-3: True or False?

There are several people who contend that nutritional deficiency might be the cause of modern disease, such as heart diseases, cancer, and depression. John Finnegan (1992) in his book, The Fact about Fat, noted that today's American diets are seriously deficient in key vital nutrients, such as vitamins, minerals, enzymes, proteins, and fatty acids. Lack of these nutrients is a major cause of current illness. He discussed how most Americans eat the wrong fat (saturated fat and poisonous trans-fatty acids) instead of polyunsaturated fatty acids. Studies that he looked at had found that the foremost nutrient that most Americans were deficient in is the key Omega 3 fatty acid. Finnegan argued that an inadequate intake of this vital nutrient has been clearly linked with helping cause most modern diseases. There would be, he argued, an optimal level of intake amount of N-3 fatty acid based on each individual's age, size, and weight, dietary history, present diet, quantity of cholesterol normally consumed, and quantity of cold water fish normally consumed. The amount of fatty acids must be carefully calculated by a certified
professional. According to Finnegan, there are only two main sources of Omega 3 fats: fish oils and organic Flax seed oil. Flax seed oil is the richer source of Omega 3 fats, requires less processing, tastes better, contains no toxic substances, is more stable, and is less expensive.

Although N-6 fatty acid is vital to human health, it might not be a cause of modern diseases because most Americans take in more than enough of N-6 fatty acids through consuming such oils as sunflower and safflower, nuts, and seeds. Thus, his main focus was N-3 fatty acids and he stressed that deficiency of N-3 would be a cause of modern diseases, including depression. The statements made by Finnegan (1992) were mostly based on the study of Rudin (1987).

Rudin and Felix (1987) conducted studies to assess how depletion of Omega-3 affected performance of monkeys and rats. Monkeys deprived of Omega-3 fatty acids for a period of two years developed severe psychotic-like reactions and many other illnesses and rats performed poorly on maze tests, although only after a lifetime of Omega-3 FAs depletion. Omega-3 EFAs, the most prominent EFA in the brain, is an essential nutrient of human—in fact, the one essential nutrient that distinguishes human and the primates from subprimates. Rudin et al. (1987) made a clear statement that a deficiency of the Omega-3 fat created widespread illnesses in the population similar to other conditions that previously existed like scurvy (caused by a deficiency of vitamin C) and pellagra (caused by a deficiency of vitamin B3). Rudin (1987) also specified that such nutrients as folic acid, vitamin B6, magnesium, selenium, Omega-3 EFAs, fiber, lactobacillus, and chromium may provide relief from depression. He has successfully treated schizophrenia,
depression, and other emotional disorders by using a good diet supplemented with 1 to 2
tablespoons a day of flax seed oil daily.

Rudin (1996) introduced the result of his 44-patient-pilot study in his book _Omega 3 Oil_. In the study, he had observed his patients over 1 to 2 years in early 1980’s. The result was that seven out of twelve patients had improved their mental status. His patients had been diagnosed as suffering from agoraphobia, mood disorders, and schizophrenia. Thus, he made a conclusion that Omega-3 fatty acids improved behavior, contributed to feelings of well-being, and reduced psychotic thinking. His conclusion might be questionable because it was based on his uncontrolled case studies. More carefully designed and implemented studies would be required before his conclusions could be given much credence.

**Summary**

Depression is a major problem for human beings. Many persons will at some time experience some degree of depression. Depressed people usually lose their appetites. In the most extreme cases, there are eating disorders, such as anorexia and bulimia nervosa. In these disorders, food and depression play major roles and these are often correlated each other. It is not obvious which can cause the other, depression or eating disorders, but there may be some relations between certain type of nutrients and depression. In this relationship, neurotransmitters can affect emotional states. Some researchers have suspected these relationships and they have conducted the studies to assess those relationships. The results from those studies have shown that there may be some relationships between nutrients (serum cholesterol and polyunsaturated fatty acids) and
depression. My study will focus on the relationship between n-3 fatty acids and depression. My hypothesis is that people who take in an adequate level of serum cholesterol and fatty acids will be less depressed than people with a deficit of those nutrients.
CHAPTER III
METHODOLOGY

Research Question

The purpose of this study was to examine possible relationships between fatty acids and mental distresses. Specifically, does increased intake of N-3 fatty acids to the optimal level result in better mental health?

Data

In order to assess whether there is any relationship between n-3 essential fatty acids and depression, three data sets were used. The first two sets concerned the prevalence of mental distress and consumption of fish and vegetable oils rich in n-3 fatty acid. Because the sampling procedures in these two data sources were not comparable, a third data source, which concerned population demographics in the United States, was also used in order to make data from the first two sources comparable.

The first data was from the 1994 study of Media and Markets by Simmons Market Research Bureau, Inc. This study was based on a total of 22,051 adults aged 18 years and older who had lived in the conterminous 48 states of the United States. The targeted food intakes of my study were fishes and some vegetable oils. These corresponded to food categories found in the “soup, meat, fish, poultry, condiments, dressing & sauces” section of the Simmons report: Fresh fish/shell fish, frozen fish/shell fish, pure canola oil, olive oil, pure corn oil, pure safflower oil, pure sunflower oil and heavy users (two quarts or
more). Of those oils, the main sources of n-3 are fish and canola oil (11%). Main sources of n-6 are safflower (76%), sunflower (71%), and corn oils (57%). The main source of monounsaturated fatty acids is olive oil in which 75% is monounsaturated, only 1% is n-3 FAs, and 9% is n-6 Fas (Canola Council of Canada, 1994). According to the Simmons Market Research Bureau, Inc. (1994), the standard format used in the report presents a four columns of data: A is the projected number of people, in thousands, in the cell defined by the heading and stub for the column and row concerned; B is the result of percentage this number down using the group defined by the heading as the base; C is the result of percentage this number across using the group defined by the stub as the base; and D is an index of selectivity calculated by dividing C by the across percent for the universe concerned (shown in Table 3 of the Appendix).

The prevalence of the food was displayed in separate four regions: Northwest, Midwest, South, and West. The Northwest area includes the New England division: Connecticut, Maine, Massachusetts, New Hampshire, Rhode Island, and Vermont; and the Middle Atlantic division: New Jersey, New York, and Pennsylvania. The Midwest area includes the East North Central division: Illinois, Indiana, Michigan, Ohio, and Wisconsin; and the West North Central division: Iowa, Kansas, Minnesota, Missouri, Nebraska, North Dakota, and South Dakota. The South area includes the South Atlantic division: Delaware, District of Columbia, Florida, Georgia, Maryland, North Carolina, South Carolina, Virginia, and West Virginia; and the East South Central division: Alabama, Kentucky, Mississippi, and Tennessee; and the West South Central division:
Arkansas, Louisiana, Oklahoma, and Texas. The West area includes the Mountain division: Arizona, Colorado, Idaho, Montana, Nevada, New Mexico, Utah, and Wyoming; and the Pacific division: Alaska, California, Hawaii, Oregon, and Washington.

The second data source was the report of the Behavioral Risk Factor Surveillance System (BRFSS). "The BRFSS is an ongoing, state-based, random-digit-dialed telephone survey of the noninstitutionalized U.S. population aged greater than or equal to 18 years that tracks the prevalence of key health- and safety-related behaviors and characteristics" (Cool et al, 1998, p. 1). This study assessed the prevalence of specific mental illnesses and conditions—frequent mental disorders (FMD). According to their definition, FMD is a continuous feeling down for greater than or equal to 14 out of the preceding 30 days that is often used by clinicians and clinical researchers as a marker for clinical depression and anxiety. This report included the table that showed the prevalence of FMD among noninstitutionalized adults by state, age group, and sex. However, the data was not comparable with the first data of food intake. It was shown in percent, not in actual numbers. It was shown in each state, not in the four regions. It was shown in eight different age-sex groups, not in a total population. The only way to make the first and second data comparable was to obtain the same population statistical data with the one this study used from the U.S. census and then to combine them into calculation.

The third data that was necessary to assess the potential relationship was the U.S. population estimates for regions and states by selected age groups and sex: annual time series, July 1, 1990 to July 1, 1998. The source was the population estimates program,
population division, U.S. Census Bureau, Washington, DC. The data was released into the Internet on June 15, 1999. The statistics of July 1, 1990 U.S. Population Estimates were comparable to the second data for the prevalence of mental distress. It included all 50 states and Washington D.C., all age groups (18-24, 25-44, 45-64, and over 65), and the four regions that were comparable to the second data (shown in Table 2 of the Appendix) were used in my assessment.

Data Analysis

Because the FMD prevalence data were provided for state-specific age x gender groups, whereas the Simmons Market Data represented consolidated regional consumption, it was necessary to recast the prevalence data into comparable terms. In order to make the calculating process easier and more accurate, the Statistical Program for the Social Science (SPSS), version 9 was used. The first step was to create a file in the SPSS data sheet. All necessary information from the data of the prevalence of FMD and of the U.S. population estimates was input. All 51 states were given the assigned number (1, 2, 3, or 4) based on the four regions (Northeast, Midwest, West, and South) and the eight age-sex groups were used. The second step was to calculate actual numbers of frequent mental distress. Since the data from the prevalence of FMD show only percentage of FMD, actual numbers were obtained by the following formula—total population of an age-sex group multiplied by (the percentage of the age-sex group divided by 100). This calculation was repeated until the actual numbers of all age-sex groups were obtained. The third step was to obtain percentages of a total population of each state (male, female, and total). The percentages of a total population of a state were
obtained by the following formula—the sum of the actual number of people with FMD in the state divided by the sum of the total population of the state aged over 18. The fourth step was to obtain the sums based on the regions. From the result of the fourth step, the prevalence rate of FMD in percentage was obtained by dividing the actual numbers of people with FMD in a region by the total population aged over 18 in the region. The result of these calculations are displayed in Table 1.

Further, the number of respondents to the FMD questions in each state for each age-gender group in the BRFSS study was estimated by making use of the using standard errors of the percentages reported for each group. Because the standard error of a proportion \( P \) is equal to the quantity \( P(1-P) \) divided by the square root of the sample size, the reported sample FMD percentages and standard errors could be used to calculate approximate sample sizes. The SPSS data spreadsheet was used to calculate the total number of the participants, those with FMD, and those without FMD for each of eight age by gender groups in each of the states.

Because the data available were not on based on linked individual responses, but instead came from aggregate regional or state data, analysis was not straightforward. As a first approach, the state was considered as the unit of analysis, and each state was assigned the food consumption values that were reported for its region. Correlation and Independent-sample t test were used to examine whether there would be any correlation observed between food intake and depression. To be specific, correlation was used in order to examine the correlation between the consumption of fish and oils, between food
and the prevalence of FMD, and between the prevalence based on age-sex groups; and Independent-sample t test was used to compare the means of two different samples in such combinations as Northeast and Midwest, Northeast and West, Northwest and South, Midwest and West, Midwest and South, and West and South. The two-tailed test was used examine whether the mean of one distribution would differ significantly from the mean of the other distribution at the \( p < .05 \) level.

The second approach used was to make use of inferred sample sizes to permit tests of regional differences that reflected the number of individual respondents sampled. One-way ANOVA was used to examine whether there would be any differences based on regions for each of the eight gender by age groups. Post hoc multiple comparison (using the Student Neuman Keuls procedure) were used to examine regional differences when significant overall mean differences were detected.
CHAPTER IV

RESULTS

During 1993-1996, the overall weighted prevalence of FMD was among adults was 8.6% (see Table 1). At the regional level, the overall prevalence of FMD among adults ranged from 8.0% in the Midwest to 9.2% in the West. Among males, FMD prevalence were highest in the West (8.9%) for those aged 18-24 years, and lowest in the Northeast and Midwest for those aged 65 years or older (5.4%). Region-level FMD prevalence among women was greatest in the Northeast and the West for persons aged 18-24 years (13.4%), and lowest in the West for persons aged 65 years and older (6.5%). The West region tended to show the highest prevalence of FMD, and the Midwest region the lowest prevalence of FMD, with the exception of the females aged 65 years or older (highest in the South at 7.1%, and the lowest in the West at 6.5%). Women were more likely to report FMD (10.2%) than men (6.9%). The younger the person, the more likely one was to feel depressed. The highest rates of FMD were among the 18-24 year-old group (males at 7.8%, females at 12.3%), and lowest rates were observed among those 65 years and older (5.4% of males, 6.8% of females).

From the 1994 Simmons study (see Table 3), the South region that exhibited the second lowest prevalence of FMD, tended to show the highest consumption of fish and all vegetable oils except olive oil. In fish consumption that enriches n-3 FAs, the South showed the highest consumption (fresh fish: 32.3%, frozen fish: 30.4%) and the West...
showed the lowest (fresh: 20.4%, frozen: 18.3%). In a canola oil consumption that is also
rich in n-3, the South showed the highest (32.4%) and the Northeast showed the lowest
(20.0%). The South region also showed the highest as heavy users of oil (39.9%) and the
West showed the lowest (18.4%). The Midwest region, which exhibited the lowest
prevalence of FMD, was generally second highest in the food intakes. The West region,
which exhibited the highest prevalence of FMD, was most likely to belong to the lowest
in the food intakes.

From Pearson correlation analysis in the SPSS computer program, the correlation
between fish and all vegetable oils were very high, ranged from .980 to .995 (p < .003).
This meant that the higher the fish consumption was, the higher the oil consumption was.
The correlation between food intake and FMD showed some moderate negative
correlation without significance among men and women aged greater than or equal to 65
years. Thus, there would be no conclusion drawn from the correlation between food
intake and depression.

From Independent-samples t test (treating the states as the units of analysis) there
were no significance found in such combined regions as NE and MW, NE and W, NE
and S, MW and S. However, there were a significance found in such combined regions as
MW (the lowest in FMD prevalence) and W (the highest in FMD prevalence) and W and
S (the second lowest in FMD prevalence). In a comparison between MW and W, a group
of males aged 25 to 44 showed a significance in their mean differences. Twelve states in
Midwest had a mean of 6.38% in prevalence of FMD of this age-sex group, 13 states in
West had a mean of 7.58% in the prevalence of FMD of this age-sex group, and differed significantly at the $p < .05$ ($p = .027$). In comparisons between West and South, a significance were seen in two age-sex groups: Male aged 25-44 and male aged over 65. In the Male aged 25-44 group, 13 states in West had a mean of 7.58% in the prevalence of FMD, 17 states in South had a mean of 6.58% in that of FMD, and the means differed significantly at the $p < .05$ level. In the Male aged over 65 group, 13 states in West had a mean of 4.56% in the prevalence of FMD, 17 states in South had a mean of 5.62% in that of FMD, and the means differed significantly ($p < .05$).

From the one-way ANOVA on the second data that calculated estimated sample size by using standard of errors instead of percentages of FMD prevalence, all age-sex groups showed significant ($p < .01$) regional differences except for Males aged 45 to 64 years. These were quite substantial and indicated regional differences in the prevalence of FMD. From the Post Hoc Multiple comparison (SNK), in the group of males aged 18 to 24, the West region was significantly different from the Midwest and South regions. Among the female aged 18 to 24, the West was significantly different from all other three regions. For the males aged 25 to 44, the Northeast and West were significantly different from the Midwest and South regions. For the female aged 25 to 44, the West was significantly different from the Midwest and South regions, whereas in the female aged 45 to 64, the South region was significantly different from the Midwest. Among the males aged 65 and over, the West was significantly different from the Midwest and South; and in the female aged 65 and over, the South was significantly different from the Northeast and West.
Table 1. Frequent mental distress (FMD) among adults,* by regions, age group, and sex - United States, Behavioral Risk Factor Surveillance System, 1993-1996

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Men</th>
<th></th>
<th>Women</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Area</td>
<td>% F p</td>
<td>Area</td>
<td>% F p</td>
</tr>
<tr>
<td>18-24 yrs</td>
<td>North east</td>
<td>8.1 4.97 .002</td>
<td>13.4 4.63 .003</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Midwest</td>
<td>6.8</td>
<td>11.2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>West</td>
<td>8.9</td>
<td>13.4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>South</td>
<td>7.5</td>
<td>11.7</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>7.8</td>
<td>12.3</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>25-44 yrs</th>
<th>Men</th>
<th></th>
<th>Women</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Area</td>
<td>% F p</td>
<td>Area</td>
<td>% F p</td>
</tr>
<tr>
<td></td>
<td>North east</td>
<td>7.1 8.28 .000</td>
<td>11.4 6.11 .000</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Midwest</td>
<td>6.5</td>
<td>10.5</td>
<td></td>
</tr>
<tr>
<td></td>
<td>West</td>
<td>7.6</td>
<td>11.9</td>
<td></td>
</tr>
<tr>
<td></td>
<td>South</td>
<td>7.1</td>
<td>10.8</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>7.1</td>
<td>11.1</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>45-64 yrs</th>
<th>Men</th>
<th></th>
<th>Women</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Area</td>
<td>% F p</td>
<td>Area</td>
<td>% F p</td>
</tr>
<tr>
<td></td>
<td>North east</td>
<td>6.5 2.18 NS</td>
<td>10.1 4.03 .007</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Midwest</td>
<td>6.2</td>
<td>9.7</td>
<td></td>
</tr>
<tr>
<td></td>
<td>West</td>
<td>7.6</td>
<td>10.3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>South</td>
<td>7.3</td>
<td>10.1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>6.9</td>
<td>10.0</td>
<td></td>
</tr>
</tbody>
</table>
Table 1 cont.

<table>
<thead>
<tr>
<th>Area</th>
<th>≥ 65 yrs</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>%</td>
<td>F</td>
<td>p</td>
<td>%</td>
<td>F</td>
<td>p</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Men</td>
<td></td>
<td>Men</td>
<td></td>
<td>Women</td>
<td></td>
<td>Women</td>
<td></td>
</tr>
<tr>
<td>North east</td>
<td>5.4</td>
<td>4.22</td>
<td>.006</td>
<td>6.6</td>
<td>4.24</td>
<td>.005</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Midwest</td>
<td>5.4</td>
<td>6.7</td>
<td></td>
<td>6.5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>West</td>
<td>4.7</td>
<td></td>
<td>6.5</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>South</td>
<td>5.7</td>
<td></td>
<td>7.1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>5.4</td>
<td></td>
<td>6.8</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
CHAPTER V
DISCUSSION

It was hypothesized that there would be a relationship between food consumption and the prevalence of depression. The result showed the region—West—with the highest rate of FMD had the lowest consumption of fishes and the second lowest of Canola oil. The region—South—with the second lowest in the prevalence of FMD had the highest consumption of fishes and all vegetables oils as well; it showed the highest number in the category of heavy users. The result that the South region showed the highest consumption in N-3, N-6 and other fatty acids oils should be taken into consideration because the N-3 and N-6 were irreversible and thus, too much N-6 could reduce the amount of the N-3. It could be an explanation of the reason why the South wasn’t the lowest in the prevalence of FMD. The result of Independent-samples t tests showed a significance that one age-sex group in the combination of MW and W and two age-sex groups in the combination of W and S. It indicated that the prevalence of FMD in a age-sex group in the Midwest region had significantly different prevalence rate from the age-sex group in the West. The result of ANOVA showed the significance in the regional differences of the prevalence of FMD. Four regions were significantly different from each other in terms of the prevalence of FMD. The finding of regional differences in consumption of fatty acids provides some circumstantial evidence to support the study hypothesis.
The relationships between polyunsaturated fatty acids and depression have been a new area to conduct a research and there were not many studies done. In the present study, two data were used in order to examine the association between N-3 fatty acids and the prevalence of Frequent Mental Disorders (FMD). The data from FMD was accompanied by the 1990 population estimate in U.S. Census Bureau that was used in those two studies so that they became comparable with each other. Both studies were conducted in a larger sample size (22,051 in Simmons Market and 436,107 in BRFSS) from all age-sex groups except people under age of 18 years and across the States, and those samples were randomized. Thus, their results were generalizable to the public. Each data was generalizable to the public. However, in a combination of the two studies, the results were to be questionable or might be invalid simply because those were separate studies. In addition, both studies were low in internal validity. There was no manipulation of independent variable(s). Those data were simply collected through either a self-administered questionnaire accompanied by personal interview or telephone survey. By using the interviews, the researchers might increase internal validity by controlling environmental factors or eliminating some confounding factors (e.g. obtain answers from a single person or multiple persons, see personal characteristics by interacting) compared to self-reports. However, it was not be enough to make a conclusion about internal validity. There was not a strong manipulation or control in the studies.

In the self-report measures, the participants assessed the degree to which some behaviors had occurred. There might be always a question of how honest participants were in answering the questionnaire. This measure was vulnerable to distortions by the
participants. They might try to look good or bad or to answer in a socially desirable way.
This concern would not be totally excluded in using the self-report measure, even in interviews.

The main problem in my study was the existence of so many extraneous and confounding factors that were impossible to be excluded from my assessment. I simply used two data from two previous studies that were done without intending to measure what I would like to assess—possible relationship between N-3 fatty acids and depression. For example, the data from the Simmons Market (1994) did not show how much oils or fishes the participants took in a daily basis. It gave what percentage of the total population or of the regional population used certain kinds of oils or ate fishes without specifying an amount. The data from BRFSS cited by Cool (1998) did not assess the participants’ biological/genetic factors (how likely they were depressed in terms of hereditary), environmental factors (stresses such as loss of loved ones, of jobs, and some changes in their lives), and personal characteristics. Depression could be caused by so many things as listed the above. The participants were simply asked how they felt at the time of the telephone survey. Without eliminating those factors, it would be quite difficult to single out a correlation between N-3 fatty acids and depression.

In addition, the association can be the result of some other confounding factors. Pijl, Toornvliet, Meinders, Leuven, and Kempen (1996) have stated that there are two confounding factors for the studies that focused on a relationship between serum cholesterol and depression. The first confounding factor is alcohol that is known to induce release of serotonin by platelets. The second confounding factor is plasma free
serotonin concentrations that are raised in peripheral vascular disease, probably because of platelet hyperactivity. These factors may affect the relationship between cholesterol and depression. Since fatty acids are highly correlated with cholesterol, these factors are more likely to be applicable to the target relationship for my assessment. Therefore, these confounding factors must be controlled in a future study in order to assess a genuine relationship between fatty acids and depression.

The relationship between fatty acids and depression were less studied than the relationship with cholesterol. This is quite new area to explore. Those were considered as possible problems in my study and future concerns. In order to improve the study, it would be important for researchers to eliminate as many extraneous variables and confounding factors as possible so that they would increase or strengthen the internal validity. The future study may be conducted by using either case study in a hospital setting or non-equivalent group pretest-posttest design in a general population. The former design is helpful to increase internal validity by controlling or manipulating independent variable(s) as well as extraneous or confounding factors although it will have little external validity. The latter design is helpful to gain insights into possible relationships between N-3 content nutrients and depression although it will not increase internal or external validity due to lack of randomization of sample and of manipulating independent variable(s).
APPENDIX

TABLES
<table>
<thead>
<tr>
<th>Area</th>
<th>18-24 yrs</th>
<th></th>
<th>25-44 yrs</th>
<th></th>
<th>45-64 yrs</th>
<th></th>
<th>&gt;=65 yrs</th>
<th></th>
<th>Total</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Men</td>
<td>Women</td>
<td>Men</td>
<td>Women</td>
<td>Men</td>
<td>Women</td>
<td>Men</td>
<td>Women</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northeast</td>
<td>2741653</td>
<td>2718835</td>
<td>8149254</td>
<td>8390187</td>
<td>4733764</td>
<td>5221183</td>
<td>2724771</td>
<td>4248479</td>
<td>38928126</td>
<td></td>
</tr>
<tr>
<td>Midwest</td>
<td>3160506</td>
<td>3122804</td>
<td>9388727</td>
<td>9577119</td>
<td>5394953</td>
<td>5779486</td>
<td>3087595</td>
<td>4666549</td>
<td>44177739</td>
<td></td>
</tr>
<tr>
<td>West</td>
<td>3045758</td>
<td>2720284</td>
<td>9110633</td>
<td>8798600</td>
<td>4495135</td>
<td>4686512</td>
<td>2431884</td>
<td>3349704</td>
<td>38638510</td>
<td></td>
</tr>
<tr>
<td>South</td>
<td>4729149</td>
<td>4591339</td>
<td>13593504</td>
<td>13907885</td>
<td>7627049</td>
<td>8345921</td>
<td>4320591</td>
<td>6409754</td>
<td>63525192</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>13677066</td>
<td>13153262</td>
<td>40242118</td>
<td>40673791</td>
<td>22250901</td>
<td>24033102</td>
<td>12564841</td>
<td>18674486</td>
<td>185269567</td>
<td></td>
</tr>
</tbody>
</table>
TABLE 3. Food Intake by Simmons Market Research Bureau, 1994

<table>
<thead>
<tr>
<th>Area</th>
<th>Fresh fish/shell fish</th>
<th>Frozen fish/shell fish</th>
<th>Canola oil</th>
<th>Olive oil</th>
<th>Corn oil</th>
<th>Safflower oil</th>
<th>Sunflower oil</th>
<th>Heavy users</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>'000 Down Across Index</td>
<td>'000 Down Across Index</td>
<td>'000 Down Across Index</td>
<td>'000 Down Across Index</td>
<td>'000 Down Across Index</td>
<td>'000 Down Across Index</td>
<td>'000 Down Across Index</td>
<td></td>
</tr>
<tr>
<td>North east</td>
<td>8937 26.7 38.6 134</td>
<td>4852 22.1 21.0 111</td>
<td>3873 20.0 26.7 100</td>
<td>7475 29.7 32.3 149</td>
<td>4200 21.0 18.1 105</td>
<td>1539 20.0 6.6 100</td>
<td>1356 20.3 5.9 102</td>
<td>7253 20.6 31.3 103</td>
</tr>
<tr>
<td>Midwest</td>
<td>6932 20.7 24.7 85</td>
<td>6380 29.1 22.7 120</td>
<td>4847 25.1 17.2 103</td>
<td>5339 21.2 19.0 87</td>
<td>5088 25.4 18.1 105</td>
<td>1496 19.4 5.3 80</td>
<td>1522 22.8 5.4 94</td>
<td>7450 21.1 26.5 87</td>
</tr>
<tr>
<td>South</td>
<td>10809 32.3 26.1 90</td>
<td>6659 30.4 16.1 85</td>
<td>6260 32.4 15.1 90</td>
<td>6950 27.6 16.8 77</td>
<td>7244 36.2 17.5 101</td>
<td>2597 33.7 6.3 94</td>
<td>2514 37.7 6.1 106</td>
<td>14056 39.9 33.9 112</td>
</tr>
<tr>
<td>West</td>
<td>6831 20.4 29.5 102</td>
<td>4018 18.3 17.3 92</td>
<td>4367 22.6 18.8 113</td>
<td>5434 21.6 23.4 108</td>
<td>3479 17.4 15.0 87</td>
<td>2076 26.9 9.0 135</td>
<td>1270 19.1 5.5 95</td>
<td>6476 18.4 27.9 92</td>
</tr>
<tr>
<td>Total</td>
<td>33508100.0 28.9 100</td>
<td>21908100.0 18.9 100</td>
<td>19347100.0 16.7 100</td>
<td>25197100.0 21.7 100</td>
<td>20010100.0 17.3 100</td>
<td>7708100.0 6.7 100</td>
<td>6662100.0 5.7 100</td>
<td>35235100.0 30.4 100</td>
</tr>
</tbody>
</table>
Table 3 cont.

<table>
<thead>
<tr>
<th>Ages</th>
<th>Fresh fish/shell fish</th>
<th>Frozen fish/shell fish</th>
<th>Pure Canola oil</th>
<th>Olive oil</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
</tr>
<tr>
<td>'000</td>
<td>Down</td>
<td>Across Index</td>
<td>'000</td>
<td>Down</td>
</tr>
<tr>
<td>18-24</td>
<td>1908</td>
<td>5.7</td>
<td>21.8</td>
<td>75</td>
</tr>
<tr>
<td>25-44</td>
<td>16024</td>
<td>47.8</td>
<td>30.6</td>
<td>106</td>
</tr>
<tr>
<td>45-64</td>
<td>10155</td>
<td>30.3</td>
<td>30.6</td>
<td>106</td>
</tr>
<tr>
<td>≥65</td>
<td>5421</td>
<td>16.2</td>
<td>25.2</td>
<td>87</td>
</tr>
<tr>
<td>Total</td>
<td>33508</td>
<td>100.0</td>
<td>28.9</td>
<td>100</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Corn oil</th>
<th>Safflower oil</th>
<th>Sunflower oil</th>
<th>Heavy users</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>A</td>
</tr>
<tr>
<td></td>
<td>'000</td>
<td>Down</td>
<td>Across Index</td>
<td>'000</td>
</tr>
<tr>
<td>18-24</td>
<td>1132</td>
<td>5.7</td>
<td>12.9</td>
<td>75</td>
</tr>
<tr>
<td>25-44</td>
<td>8173</td>
<td>40.8</td>
<td>15.6</td>
<td>90</td>
</tr>
<tr>
<td>45-64</td>
<td>6360</td>
<td>31.8</td>
<td>19.2</td>
<td>111</td>
</tr>
<tr>
<td>≥65</td>
<td>4345</td>
<td>21.7</td>
<td>20.2</td>
<td>117</td>
</tr>
<tr>
<td>Total</td>
<td>20010</td>
<td>100.0</td>
<td>17.3</td>
<td>100</td>
</tr>
</tbody>
</table>
REFERENCES


http://www.cdc.gov/epo/mmwr/preview/mmwrhtml/00052469.htm


